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Infectious Bursal Disease(GUMBORO Disease) in Chickens

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Abstract: Infectious Bursal Disease (IBD) is an immune system disease which is caused by a virus which is a member of the genus *Avibirnavirus* of the family *Birnaviridae*. The objective of this paper is to review available information on the etiology, epidemiology, economic significance, diagnosis, control and prevention method of IBD. Although turkeys, ducks, guinea fowl and ostriches may be infected, clinical disease occurs solely in chickens. Only young birds are clinically affected. Severe acute diseases of 3-6-week-old birds are associated with high mortality, but a less acute or subclinical disease is common in 0-3-week-old birds. This can cause secondary problems due to the effect of the virus on the bursa of fabricius. IBD virus causes lymphoid depletion of the bursa and if this occurs in the first 2 weeks of life, significant depression of the humoral antibody response may result. Two serotypes of IBDV are recognized; these are designated serotypes 1 and 2. Clinical disease has been associated with only serotype 1 and all commercial vaccines are prepared against this serotype. Very virulent strains of classical serotype 1 are now common and are causing serious disease in many countries. Clinical disease due to infection with the IBDV, also known as Gumboro disease, can usually diagnose by a combination of characteristic clinical signs and post mortem lesions. An effective IBD prevention and control program must involve an effective breeder vaccination program, an effective bio-security program and effective broiler vaccination programs are recommended to prevent IBD.

Key words: Birds · Gumboro · Infectious bursal disease · Immunity

INTRODUCTION

Poultry production is an important agricultural activity for most rural communities in Africa. It provides rural house hold with scarce animal protein in the form of meat and eggs as well as reliable source of petty cash [1]. There are about 56.5million of poultry species in Ethiopia. Local chicken constitute about 99% total poultry population in the small scale rural farm, however losses due to chicken mortality that occurs in different age group is very high (61%)[2].

Despite these facts, the contribution of poultry production to small holder farmer and country economy is still restricted by various factors like low input of feeding, poor management, infectious disease, lack of appropriate selection and breeding practice [2]. However, attempt are underway to enhance chicken productivity and their contribution through importing exotic chickens and also by cross breeding and distributing improved breeds to poor farmer living in rural part of Ethiopia [3].

Infectious bursal disease is the major health and production constrain of young chicken. IBD is acute highly contagious globally occurring viral poultry disease. The causal agent belongs genus *Avibirnavirus* offamily *Birnaviridae*. High pathogenic strain of IBDV is known to cause 100% morbidity and mortality of 20-30%. IBDV is present in two clinical form. Acute on set high mortality in chicken up to 20%,usually in bird around 3-4 weeks old. Immune- suppression disease as the result of infection at early age, predisposing bird to secondary infection. Old birds show the subclinical form of IBD depending on the strain and amount of virus, age and breed of bird. A previous study in Ethiopia indicating that mortality rate of IBD range from 45-50%. The overall prevalence of IBD antibody recorded in different part of country and poultry production system reached up to 93.3% [3,4].

The disease is characterized mainly by the swollen and hyperemic bursa of fabricious during acute stage (3 to 4 days post infection) and then severe atrophy of the

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organ. Accompanying symptoms include white watery droppings, accumulation of urate in the urinary structures and severe depression [5]. The disease is transmitted through water, feed and droppings [6]. The virus has predilection for lymphoid tissue special target organ being the bursa and also can be isolated from the thymus, spleen and bone marrow. Besides the loss due to mortality and morbidity, immunosuppression is a very important problem associated with IBD infection [7]. The IBD virus destroys lymphocytes and macrophages as a result cripples the immune system with marked immunosuppressive effect leading to vaccination failures and concurrent infections [4].

The clinical form of the disease is less importance now a day, occurs in chickens over weeks of age when the bursa are well developed. The greatest economic losses are due to sub clinical disease in chicks fromone to twenty one days of age. At this stage the virus impairs the immune response and renders the chick susceptible to various infections. The effects of late infection from three to ten or more weeks of age result in the clinical disease [8].

Pathological lesions include hemorrhages in leg muscles usually of pin point type. Swollen kidneys filled with white ‘urate deposits;uraters filled with the white substance and accumulation of white stony substance in cloaca [9]. The expected lesions of swollen and hemorrhagic or atrophied bursas were less frequent. Histological studies of the affected bursa revealed congestion of the vascular layer, lymphoid cell necrosis, hyperplasia of the reticuloendothelia cells and interfollicular tissue [6]. Therefore, the major objective of this review paper is to assess on infectious bursal disease(IBD) and evaluate the economic significance.

Infectious Bursal Disease (Gumboro Disease): Infectious bursal disease is an acute and highly contagious disease of young chicken which is caused by *infectious bursal disease virus* (IBDV). The causal agent was first isolated in Gumboro, Delawer(in United States of America [USA]), and the disease was originally known as Gumboro disease [10].

The Infectious bursal disease virus replicates immature B-lymphocyte in BF leading to reduced immologic responsive of chicken [8]. Disease characterized by sudden onset short course and extensive destruction of lymphocyte particularly in BF(bursa of fabricius) and other lymphoid organ [11]. It considered as AIDS of chicken since it severely affected chicken immune system [12].

Etiology: Infectious bursal disease (IBD) caused a virus of genus *Avibirnaviirus* of family Birnaviridae[9, 13-15]. Birnaviridae are contains two segments of linear, double stranded RNA. It replicates in cytoplasm of host cell and involve a virion-associated RNA dependent and RNA polymerase. Family *Birnaviridae* contains the genera which affect chicken, fish and insect [14].

Infectious bursal disease particles have a non-enveloped, icosahedra capsid with diameter of about 60nm. Formally the virus of infectious bursal disease virus (IBDV) was improperly classified as *Picorn*a or *Reovirus*. However, the known biological and structural properties of IBDV didn’t allow its classification in one of the established taxonomic groups[13, 16]. Characterization of viral genome as bi-segmented double strand RNAMuller *et al.* [17] allowed placing IBDV into a new family of virus the *Birnaviridae.* Two serotypes (1and 2) of IBDV have been described only serotype 1 appears to be pathogenic chicken [18].

Physicochemical properties of infectious bursal disease-The virus of IBDV is very stable and resistant to variety of chemical and disinfectant. It is resistant for treatment with chloroform and ether, an affected by ph 2 but inactivated ph 12.The virus is unaffected by exposure for 1 hour at 0.5% to 30% phenol and 0.125% trimersal. Virus infectivity was markedly reduced when exposed to 0.5%formalin for 6 hours. The virus also known for its resistant against different chemical like phenolic derivative and a quaternary ammonium compound, but iodine complex has a deleterious effect on virus. IBDV also heat stable, viable after treatment at 56 °C for 5 hours[8].

Epidemiology: Incidence and distribution-Infectious bursal or Gumboro disease is a highly contagious disease of young chicken that can be associated with high morbidity and mortality. Subclinical infection with IBDV causes severe lymphocyte depletion in BF, resulting in immune suppression [19]. IBD occurs worldwide in major poultry production area and 80% of member countries of OIE report the occurrence of acute clinical case (vv IBDV)[20]. Since 1986, Europe has experienced the emergency of vv strain of IBDV, which can cause up to 70% flock mortality in laying pullet [13]. However, vv IBDV can establish infection in face of level of MDV that were previously protect against "classical" strain, meanwhile, vv IBDV infection also have been observed in Africa, Asia and only recently in South America [21].

The first report of a specific disease affecting the BF in chickens was made by Cosgrove in 1962. The first cases were observed in the area of Gumboro, in Delaware (United States of America [USA]), which is the origin of the name, although the terms 'IBD' or 'infectious bursitis' are more accurate descriptions. Between 1960 and 1964, the disease affected most regions of the USA and reached Europe in the years 1962 to 1971[22]. From 1966 to1974, the disease was identified in the Middle East, southern and western Africa and India [13]. The disease is currently an international problem:95% of the 65 countries that responded to a survey conducted by World organization for Animal Health (OIE) in 1995 declared cases of infection [20].

Host range-Only chickens develop IBD after infection by serotype 1 viruses. Turkeys may be asymptomatic carriers of serotype 2 and at times of serotype 1 viruses whose pathogenicity for turkeys is ill defined. The duck can also be an asymptomatic carrier of serotype 1 viruses. Anti-IBDV antibodies have been detected in guinea-fowl, common pheasants,and ostriches,which have also been demonstrated to carry serotype 2 viruses. Neutralizing or precipitating antibodies have been detected in various species of wild duck, goose, puffin,and penguin, which may mean that wild birds act as reservoirs or vectors [13].

Transmission-Only horizontal transmission has been described, with healthy subjects being infected by oral or respiratory pathway and conjunctiva. The infected subject excretes the virus in faces as early as 48hours after infection and may transmit the disease by contact over a sixteen-day period. The Possibility of persistent infection in recovered animal has not been recovered. The disease transmitted by direct contact with excreting subject, or by indirect contact with any an animate, or inanimate (farm, staff, animal) contaminated vectors. The houses in which outbreak has been occurred remain infectious for 54 to 122 days [13]. The virus is not egg transmitted but can survive on the eggshell surface [23]. The role of wild birds and rodents are uncertain, but they may act as mechanical carriers. Mealworms have been implicated as reservoirs; infected chickens continue to excrete the virus in their feces for up to 2 weeks after infection [24].

Morbidity and Mortality-In infected flocks, morbidity is high, with up to 100% serological conversion, after infection, mortality is variable. Until 1987, the field strains isolated was of low virulence and caused only 1% to 2% of specific mortality. However, since1987 an increase in specific mortality has been described indifferent parts of the world. In the USA, new strains responsible for up to 5% of specific mortality were described. These hyper virulent field strains caused up to 100% mortality in specific-pathogen free (SPF) chicken. Generally the mortality rate in fully susceptible flock is 20-30% [20].

Pathogenesis: The virus affect lymphoid tissue causing destruction of B lymphocyte cell within the BF, the spleen and cecal tonsil-lymphocyte relatively unaffected, The most common route of infection is oral, but conjunctiva and respiratory route may also be important. Four to five hours after oral infection virus can be detected in macrophases and lymphoid cell in the cecum, duodenum, jejunum and kuppfer cell of the liver. The bursa is infected via the blood stream and by 11 hour many cell in this organ contain antigen. A viremia follow when the virus infect other organ including spleen, the harderian gland and the thymus lymphocyte and their precursor appear to viral antigen can be found in the bursa up to 14 days post infection [25].

In some birds the kidneys appear swollen and may contain urate deposit and cell debris which is probably result of blockage of ureters by severely swollen bursa. The cause of muscle hemorrhage is unknown. Bursa depletion as the result of virulent IBD virus infection in early life can result in impaired immune responses to antigen and the response to IBD virus itself. Although there are reports indicating that infection as late as 4 weeks of age results poor response to certain antigen. This not all the cases and the severity of infection and whether or not maternal derived antibodies (MDA) modified the disease could be important. The consequence of immunosuppression is lowered resistance to disease and suboptimal response to vaccine given during this time [19].

Clinical Signs: Infectious bursal disease virus has short incubation period of 2-3 days and the infection generally last 5-7 days. One of the earliest sign of IBDV infection is the tendency for bird to engage in vent picking. Clinical sing are described as acute onset of depression, trembling, white and watery diarrhea, anorexia, prostration, ruffled feather,and vent feather solids with urates. In severe cases, bird became dehydrated and in terminal stages subnormal temperature and death [2].

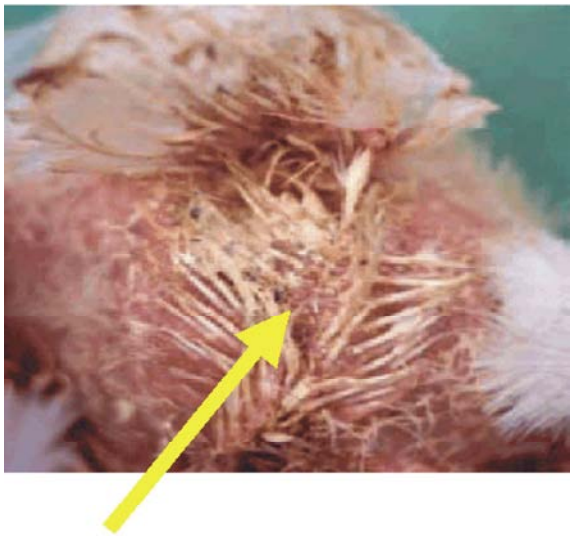

Fig. 1: The feathers around the vent are usually stained with feces containing plenty of urates
Source: [26]

Mortality commences on the third day of infection, reaches a peak by day four, then drops rapidly and the surviving chickens recover a state of apparent health after five to seven days. Disease severity depends on the age and breed sensitivity of the infected birds, the virulence of the strain and the degree of passive immunity. If the virus persists on the farm and is transmitted to successive flocks, the clinical forms of the disease appear earlier and are gradually replaced by subclinical forms. Moreover, a primary infection may also be inapparent when the viral strain is of low pathogenicity or if maternal antibodies are present [13].

Subclinical and Clinical IBD: Infectious bursal disease follow one of the two courses, depending on the age at which chicken are infected. The subclinical form disease occurs in chicken less than 3 weeks age. Chicken present no clinical sign of disease, but experience permanent and sever in immunosuppression. The reason young chicken exhibit no clinical sign of disease are not known. However, immunosuppression occurs due to damage of bursa of fabricius [25]. Typically have poor body weight and feed conversions, high mortality processing. The poor performance of chicken is due to immunosuppression caused by subclinical IBD. The clinical form of IBD usually occurs in chicken from 3-6 weeks of age. The clinical disease has a sudden onset and mortality rate in the flock increase rapidly. Clinical form of disease includes dehydration, trembling, ruffled feathers, vent picking and depression [5].

Pathology: Gross lesions-The lesions observed in bird that are common to IBDV infection include dehydration, hemorrhage in breast and leg musculature, darkened discoloration of pectoral muscles, occasional hemorrhage in thigh muscle and pectoral muscle, increasing mucus in the intestine and renal changes. In bird that are die or in advanced stage of the disease, kidneys frequently show swelling and pallor with accumulation of urates in the predominant lymphoid organ affected by IBDV[27].

As the bursa is primary target organ of the virus, it is important to understand the sequence of change while examining bird at postmortem. On 3rd day following infection bursa begins to increase in size and weight, because of accumulation of fluid (edema and more blood in the organ)[14]. By the 4th day, bursa usually is double its normal weight and size and then after begins to decrease in size. From 8th day onward, it is about one third its normal weights [26]. The bursa usually show necrotic foci (area of dead tissue) and cheesy mass is found within its lumen from fallen cell of tissue. At time small large hemorrhage on its inner surface (mucosal surface) is also seen. Sometimes wide spread hemorrhage throughout the entire bursa are present in such case, bird may pass blood in their drooping [15].

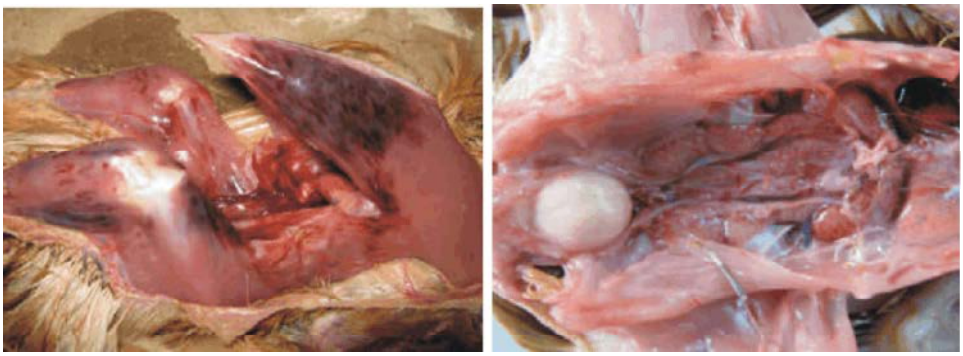

Fig. 2: Hemorrhages in the pectoral, thigh and abdominal muscles and inflamed bursa: Source [25].

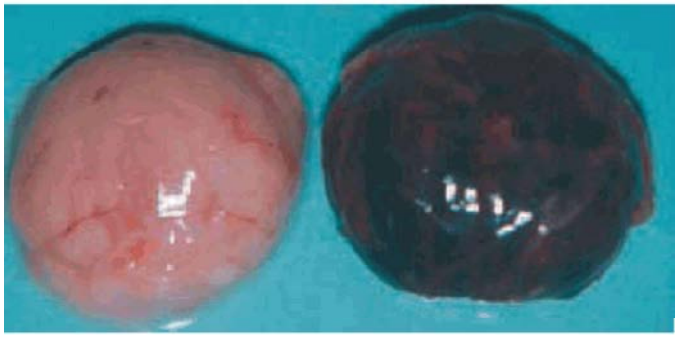

Fig. 3: Various stages of serous hemorrhagic to severe hemorrhagic inflammation of bursa Source: [26]

Moderate to severe splenomegaly with small gray foci uniformly distributed on the surface has been reported. Occasionally, petechial hemorrhages have been in the mucosa at the junction of Proventricules and gizzard [28].

Microscopic lesions - IBDV infection produced microscopic lesion primarily in the lymphoid tissue that cloacal bursa, spleen, thymus, cecal tonsil and harderian gland. Degeneration and necrosis of B-lymphocyte in the medullary region of bursal follicles is apparent within one day of exposure. Depleted lymphocyte are quickly replaced by hetrophil and hyperplasic reticulo-endothelial (RE) cells. By 3 or 4 infection, IBDV associated lesion are visible within all bursal follicles [5].

At this time, infections with classic IBDV strains have caused an inflammation response marked by severe edema, hetrophil infiltration and hyperemia in the bursa. Inflammations diminish by day 4 post infection and as necrotic debris is cleared by phagocytosis, cystic cavities develop in the medullary area of the lymphoid follicles. Necrosis and infiltration of hetrophil and plasma cell occur within the follicle, as well as, the interfollicular connective tissue. In addition, fibroplasias the interfollicular connective tissue may appear and the surface epithelium of bursa involutes and abnormal [29]. Proliferation of bursa epithelial layer generates a glandular structure of columnar epithelium microscopic lesion caused by variant strains are characterized by extensive follicular lymphoid depletion and rapid atrophy of the cloacal bursa [30].

Diagnosis: it involves consideration of flock history, clinical sign and post mortem lesion [17]. Pathological change observed at the bursa of fabricius is characteristic and histophatological investigations combined with the demonstration of viral antigen by immunohistochemistry confirm an IBDV infection.. Viral antigen can be demonstrated by agar gel precipitin assay or by antigen capture enzyme linked immunosorbent assay (AC-ELISA) [8].

AC-ELISA allow the identification of vv IBDV to demonstrate the presence of IBDV specific anti bodies. ELISA system are commercially available. The virus neutralization assay is the only serological test, which can reliably differentiate IBDV isolate in to antigenic serotype subtype [31]

Now a day, reverse transcription-polymerase chain reaction (RT-PCR) is a molecular tool frequently applied in IBDV diagnosis. RT-PCR in combination with restriction enzyme analysis allows rapid identification of vv IBDV [32].

Different Diagnosis: The sudden onset, high morbidity, ruffled feathers and droopy appreance of the bird in initial disease outbreaks are suggested of an acute outbreak of coccidiosis. In some cases there is blood in drooping that would lead one to suspect coccidiosis. The elarged muscular hemorrhages and elarged edematous or hemorrhagic cloacal bursas would suggest IBD [10].

Bird that from acute IBD may show an acute nephrosis, because many other condition that may cause nephrosis and inconsistency of kidney lesion, such lesion should be sufficient cause for diagnosis for IBD. Again involvement of cloacal bursa usually will distinguish IBD from other nephrosis causing condition. Maker's (cause bursal atrophy, but nerve lesion are very distinct). Hemorrhage syndrome (cause bursal muscular mucosal hemorrhage, but with no bursal lesion) is the usual manifestation of the diseases [27].

Treatment: There is no specific therapy for the disease. Facilitate the access to water to prevent dehydration. As with every disease optimize climate and reduce stress to a minimum. Use of antibiotics can sometimes be advisable to limit the impact of secondary infections [33].

Prevention and Control: Due to stable nature of virus and large amount of execrated following infection, it is practically impossible to remove all source of infection

once a rearing site has been contaminated. There is evidence, however, that through cleaning and disinfection of houses between flock and practice of all- in all- out management reduced the challenge of virus. It may also delay challenge thus allowing time for vaccine to induced immunity. Formaldehyde and iodophors have been shown to be effective disinfectant [25].

A good biosecurity, cleaning and disinfection and good chick source are important to insure that the chicken would be to face any diseases. IBDV is highly infectious and very resistant to inactivation therefore, despite strict hygienic measures; vaccination is inevitable under high infection pressure and mandatory to protect chickens against infection during the first week after hatch [12].

To induce high titer of maternally derived antibodies that persist over the whole laying period, layer are vaccinated with inactivated vaccine, after hatching chicken immunized with live vaccine. The titer may vary considerably with in a flock and are vaccination may be necessary. It has also to be taken in to consideration that vv IBDV will break through the immunity provided by highly attenuated vaccine strain on the other side, it is well known that less attenuated strain may cause lesion in the bursa follicles and, thus immunosuppression even in vaccinated chicken [13].

Economic Significance: IBD is important immunosuppressive viral disease of chickens. It has been described throughout the world and its social- economic significance is recognized worldwide [31]. The economic loss due to this disease could be due to direct mortality of chicken during acute lethal course or by the immune-suppression. Most the economic devastation associated with IBD is due to its immunosuppressive effect that leads to poor vaccination response, secondary bacterial, viral, protozoan infection and poor performance and poor economic return [13].

CONCLUSION AND RECOMMENDATIONS

Infectious bursal disease is a contagious chicken disease caused by a virus that destroys lymphocytes, primarily in the bursa of Fabricius, but also in other organs of the immune system, like the thymus, the spleen and the caecal tonsils. The result is a marked immune-suppressive effect causing increased susceptibility to other diseases and impaired response to many vaccinations.Effective control of IBD in commercial broilers requires that field virus exposure is reduced by proper clean-up and disinfection between flocks and that traffic (people, equipment and vehicles) onto the farm be controlled. The development and enforcement of a comprehensive biosecurity program is the most important factor in limiting losses due to IBD. Iodophors and formaldehyde compounds have been shown to be effective for disinfection of contaminated premises. Efforts at biosecurity (cleaning, disinfecting, traffic control) must be continually practices between flock.

Based on the above conclusion the following recommendations are forwarded:

- Following outbreak, the farm should be depopulated and completely cleaned and disinfected.
- Following disinfection using, iodophors and formalin and poultry house should be vacant for at least 6 month.
- The chicken should be vaccinated to prevent the disease especially important is the immunization of breeder flock inorder to providing maternal antibodies to their progeny.
- Very strict restriction should be imposed for entry of vehicle, equipment and visitors t to the farm.

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